Comment

Our data contrast with those of Dow *et al*[2] in that they suggest that added testosterone is beneficial in alleviating psychosexual symptoms. One reason for the difference may be the selection process: Dow *et al* studied an unselected group of patients attending their menopause clinic while we chose patients in whom oral oestradiol had failed to relieve psychosexual symptoms. Perhaps only in this group is testosterone specifically effective. No significant side effects were experienced.

We are grateful to Peter Dennis, Ken Outch, Nick Balazs, and Mohan Bangah for hormone and lipid assays, to Jill Volfsbergs for secretarial help, and to Robyn Redman for the artwork. Organon Australia kindly donated the implants.

1 Burger HG, Hailes J, Menelaus M, Nelson J, Hudson B, Balazs N. The management of persistent symptoms with oestradiol-testosterone implants: clinical, lipid and hormonal results. *Maturitas* 1984;6:351-8.
2 Dow MGT, Hart DM, Forrest CA. Hormonal treatments of sexual unresponsiveness in postmenopausal women. *Br J Obstet Gynaecol* 1983;90:361-6.

(Accepted 23 January 1987)

Prince Henry's Hospital, Melbourne, Australia 3004
HENRY BURGER, MD, FRACP, director, medical research centre and department of endocrinology
JEAN HAILES, MB, BS, clinical assistant, menopause clinic
JOY NELSON, MB, BS, clinical assistant, menopause clinic

Endocrine Unit, Royal Women's Hospital, Melbourne, Australia
MARGARET MENELAUS, MB, BS, clinical assistant

Correspondence to: Dr Burger.

Medical leeches as sources of wound infection

Leeches are used in plastic surgery to help relieve venous congestion after microsurgery: they ingest blood until satiated and then detach themselves. Owing to the anticoagulant properties of the animals' saliva the puncture wound continues to bleed. We report six cases in which use of leeches was followed by wound infections due to *Aeromonas hydrophila*.

Patients, methods, and results

We use about 100 leeches (an average of 10 per patient) each year. During the past three years six wound infections caused by *A hydrophila* developed in patients on whom leeches had been used; this represents an infection rate of 20%. The table summarises details of the six cases.

Comment

Whitlock *et al*[1] suggested that the medicinal leech (*Hirudo medicinalis*) was a potential source of infection when used in plastic and reconstructive surgery because it carries *A hydrophila* within its gut. The leech has no proteolytic gut enzymes and relies on the bacterium to digest blood. The organism has also been isolated from the anterior and posterior suckers of leeches and from the mucous trail and water in which they were kept.[1] *A hydrophila* has been implicated in three types of infection. It has been reported to be the causative organism in 2% of patients with diarrhoea,[2] and infections may occur after injuries sustained while swimming in contaminated water[3] and in immunocompromised patients.[4] The organism is occasionally carried in faeces.[2]

Although infection after the use of leeches has been reported only once previously,[5] our findings suggest that leeches may be an important cause of wound infections. These infections are generally characterised by the onset of inflammation and suppuration over 24 hours accompanied by moderate fever and leucocytosis. The proteolytic action of the bacterium may explain its effect on muscle; in case 5 a graft of muscle was destroyed by infection. The infection responded to antibiotic treatment in all but one case, in which it settled spontaneously before sensitivities were known. Surgical drainage and debridement were used to remove necrotic material.

Despite our findings leeches will remain useful in plastic and reconstructive surgery to treat venous congestion. When wound infection occurs after their application aeromonas should be looked for carefully. All but one of the strains isolated from our patients were resistant to penicillin and ampicillin; antibiotic sensitivity tests should always be performed. To treat an aeromonas infection an antibiotic resistant to β lactamases (for example, Augmentin) should be given orally or a cephalosporin (for example, cefuroxime or cephradine) intravenously.

Data on patients with A hydrophila wound infections

Case No	Date of operation	Indication	Surgical procedure	Time to positive swab (days)*	Clinical picture	Sensitivity to ampicillin/ penicillin	Treatment	Outcome
1	May 1983	Carcinoma of nasal septum	Forehead flap to upper lip	26†	Wound discharge	−	Cefuroxime	Settled
2	Aug 1983	Traumatic amputation of right thumb tip	Cross finger flap	2	Tenosynovitis	−	Augmentin	Settled
3	Sept 1984	Traumatic amputation of right hand	Microsurgical repair	11	Pin site, wound discharge	−	Debridement	Settled
4	Aug 1986	Midtarsal fracture of left foot	Instep flap to bare bone	4	Wound discharge and skin necrosis	−	Cefuroxime, debridement	Settled
5	Sept 1986	Cranioplasty	Microsurgical latissimus dorsi free tissue transfer	1	Infected haematoma	+	Amoxycillin, debridement	Settled
6	Nov 1986	Degloving injury of right hand	Microsurgical repair	7	Wound discharge and skin necrosis	−	Cefuroxime, debridement	Settled

*Time from first application of leeches to date of first positive swab result.
†Patient underwent staged procedures with leeches applied after each stage.

1 Whitlock MR, O'Hare PM, Sanders R, Morrow NC. The medicinal leech and its use in plastic surgery: a possible cause of infection. *Br J Plast Surg* 1983;36:240-4.
2 Millership SE, Curnow SR, Chattopadhyay B. Faecal carriage rate of Aeromonas hydrophila. *J Clin Pathol* 1983;36:920-3.
3 Rosenthal SG, Bernhardt HE, Phillips JA. Aeromonas hydrophila wound infection. *Plast Reconstr Surg* 1974;53:77-9.
4 Bulger RJ, Sherris JC. The clinical significance of Aeromonas hydrophila: a report of two cases. *Arch Intern Med* 1966;118:562-4.
5 Dickson WA, Boothman P, Hare K. An unusual source of hospital wound infection. *Br Med J* 1984;289:1727-8.

(Accepted 19 January 1987)

Department of Plastic and Jaw Surgery and Department of Medical Microbiology, Frenchay Hospital, Bristol BS16 1LE
N S G MERCER, FRCS, registrar in plastic surgery
DEBORAH M BEERE, MB, BS, senior registrar in medical microbiology
A J BORNEMISZA, FIMLS, senior medical laboratory scientific officer
P THOMAS, FIMLS, chief medical laboratory scientific officer

Correspondence to: Mr Mercer.

Primary myelodysplastic syndrome and cancer

Myelodysplastic syndrome is a preleukaemic clonal abnormality of haemopoietic stem cells.[1,2] We have looked at the occurrence of non-haematological malignancy in 138 patients with this condition, to find out whether or not they have an increased risk of developing other cancers either before or after myelodysplastic syndrome has been diagnosed.

Methods and results

Between October 1982 and May 1986 myelodysplastic syndrome (defined by conventional criteria[1,2]) was diagnosed in 138 patients. No patient with coexistent

evidence of renal or hepatic failure or malignancy at the time of diagnosis was included in this study. Evidence of previous malignant neoplastic disease (ICD codes 140-208) was sought in all cases and, when found, the dates of the two diagnoses confirmed. All patients were followed up at least every six months until death or 31 May 1986, and any subsequent diagnosis of malignant neoplasm was recorded.

The incidence of malignant neoplasms is recorded by cancer registries and regularly reported nationally, but prevalence is not commonly available in routine health statistical data. Prevalence by sex and by five year age groups in Wales as at 31 December 1983 was provided by the Welsh Cancer Registry (Welsh Office, personal communication). The prevalence of previously diagnosed malignant neoplasms in myelodysplastic patients was compared with the expectation for the Welsh population by sex and five year age group. The incidence of malignant neoplasms diagnosed after the myelodysplasia in this series of patients was compared with the expectation using data compiled in England and Wales in 1982[3] and the numbers of person years at risk in each sex and five year age group. Skin cancers were considered separately because of the possible weakness of incidence and prevalence data.

The prevalence estimate of malignant neoplasms in a population with the age and sex distribution of this series of myelodysplastic patients was 6·12 (Welsh Office, personal communication), which was not statistically different from the four observed cases. When skin cancers were included on both sides of the comparison the expected figure increased by 2·08 and the observed by 3, so that, again, there was no significant difference. After myelodysplastic syndrome had been diagnosed follow up resulted in 92 male and 85 female person years at risk. The table summarises the number of cancers that would have been expected on the basis of the person years at risk in each age group and the incidence of malignant neoplasms in England and Wales in 1982 (Welsh Office, personal communication). While 2·45 cancers would have been expected, 7 were found. The Poisson probability of such a difference occurring by chance was 0·004.

Malignant neoplasm diagnosed after myelodysplastic syndrome in 138 patients

	Men			Women		
Age group	Person years at risk	Expected cancers*	Observed cancers	Person years at risk	Expected cancers*	Observed cancers
≤59	26·3	0·11	0	21·5	0·10	0
60-64	12·0	0·12	1	5·2	0·04	0
65-69	16·7	0·27	0	9·4	0·09	0
70-74	11·2	0·25	1	28·1	0·34	1
75-79	8·6	0·24	1	12·6	0·18	2
≥80	16·7	0·54	0	8·3	0·15	1
All ages	91·5	1·54	3	85·1	0·91	4

*Expected number derived from total person years at risk in each age group and incidence of malignant neoplasms in England and Wales in 1982.[3]

Comment

In this group of 138 patients with myelodysplastic syndromes non-haematological malignancy occurring during follow up occurred at 2·9 times the expected rate. The exclusion of those patients in whom preleukaemia and a non-haematological malignancy were diagnosed at the same time probably led to an underestimate of the coincidence of these conditions. Our findings suggest that patients with myelodysplastic syndrome may have a predisposition to develop other cancers later. A higher incidence of other cancers occurring before the diagnosis of myelodysplasia may have been obscured by death before the haematological abnormality was identified. We can only speculate on the reason for a higher than expected coincidence of preleukaemia and other cancers, though both hereditary and environmental causes may be implicated.[4]

1 Jacobs A. Myelodysplastic syndromes: pathogenesis, functional abnormalities and clinical implications. *J Clin Pathol* 1985;38:1201-7.
2 Galton DAG. The myelodysplastic syndromes. Part 1. What are they? Part II. Classification. *Scand J Haematol* 1986;36(suppl 45):11-20.
3 Office of Population Censuses and Surveys. *Cancer registration England and Wales 1982*. MBI.14. London: HMSO, 1985.
4 Jacobs A. Human preleukaemia: Do we have a model? *Br J Cancer* 1987;55:1.

(Accepted 23 January 1987)

Department of Haematology, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN
R E CLARK, MD, MRCP, senior registrar
H E PAYNE, MB, BS, leukaemia research fund fellow
A JACOBS, MD, FRCPATH, professor

Department of Epidemiology and Community Medicine, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN
R R WEST, PHD, MA, senior lecturer

Correspondence to: Professor Jacobs.

Chronic lymphocytic leukaemia contemporaneous with HIV infection

People infected with the human immunodeficiency virus (HIV) have a range of cell counts ranging from lymphopenic to absolute lymphocytosis.[1] The lymphocyte changes mainly affect T cells and can be varyingly attributed to the infection of T helper lymphocytes, to other microbial infections, or to exogenous compounds affecting T cell subsets differentially. B lymphocyte dysfunction and lymphocytosis attributable to polyclonal B cell stimulation by concomitant Epstein-Barr virus infection and to loss of T cell regulation of B lymphocytes has also been documented. We describe here a man with an idiopathic lymphocytosis and evidence of contemporaneous infection with HIV who was diagnosed as having chronic lymphocytic leukaemia.

Patient, methods, and results

A 50 year old man presented in July 1984 with a five week history of malaise, fevers, abdominal discomfort, and weight loss. He had no lymphadenopathy or splenomegaly and the liver edge was just palpable but not tender. Blood investigations showed normal haemoglobin and platelet count with a white cell count of 25×10^9/l, 85% of the cells being mature small lymphocytes. Marrow aspirate examination showed a light infiltration with similar lymphocytes. No paraprotein was present and immunoglobulin concentrations were normal. He disclosed that he was an active homosexual and that he had returned from holiday in Haiti in April 1984. He was negative for hepatitis B surface antigen and for HIV antibodies. His symptoms resolved spontaneously and he was discharged to be followed up.

He remained physically well apart from suffering several boils in the axillae and on the thighs, culture from which showed *Staphylococcus aureus*. In July 1985 he was noted to have enzyme changes characteristic of liver damage, at which time serological tests for hepatitis B became positive; antibodies to HIV were also found and confirmed by Western blotting. He developed no lymphadenopathy or hepatosplenomegaly.

Purified peripheral blood mononuclear cells were subjected to immunophenotyping using indirect immunofluorescent staining of cell suspensions and the more sensitive alkaline phosphatase-antialkaline phosphatase method.[2] The patient's lymphocytes were incubated with mouse monoclonal antibodies to various human cluster of differentiation (CD) antigens and to other T and B cell associated antigens. The lymphocytes were also assayed in a mouse rosette test, a well established technique for showing chronic lymphocytic leukaemia B cells.

Using a human polyclonal antiserum to HIV, which in Western blot analysis detected various core and envelope antigens of the virus, we examined the patient's cells for expression of viral antigens by an indirect immunoperoxidase method.[3] The immunophenotypic results are shown in the table.

Immunophenotypic analyses in patient with white blood count of $19\cdot2\times10^9$/l, lymphocytes 81%, absolute count $\sim15\cdot5\times10^9$/l

Monoclonal antibodies to:	Percentage positive cells	Absolute positive cell count ($\times10^9$/l)	Normal range absolute count ($\times10^9$/l)
CD3 (pan T cells)	20	3·1	(1·0-2·4)
CD4 (T helper subset)	7	1·1	(0·6-1·7)
CD8 (T cytotoxic/suppressor subset)	9	1·4	(0·2-1·0)
IgM	17	2·6	(0·04-0·4)
κ*	5	0·8	(0·04-0·28)
λ*	80	12·4	(0·02-0·19)
HLA-DR	80	12·4	(0·19-0·33)
CD5 (pan T+chronic lymphocytic leukaemia cells)	70†	11·0	
Mouse rosette	~50 rosetting cells were detected (normal range <5%)		

Anti-HIV staining of the neoplastic cells was uniformly negative.
*Anti-κ and anti-λ results were obtained by the sensitive alkaline phosphatase technique.[2]
†The result obtained with CD5 compared with CD3, CD4, and CD8 indicated that CD5 was detecting mainly B chronic lymphocytic leukaemia cells and not T cells.

Comment

The patient's lymphocytosis was mainly attributable to a population of cells which were CD5, HLA-DR, and mouse rosette positive—the predominant phenotype of B cell chronic lymphocytic leukaemia. The neoplastic nature of the cells was confirmed by the expression of monotypic λ light chains.[2]

According to the criteria of the Centers for Disease Control this patient did not have the acquired immune deficiency syndrome (AIDS), and he continued to remain well. His chronic lymphocytic leukaemia seemed to be non-symptomatic and non-progressive. If it were to become symptomatic the problem would arise of what treatment, if any, to give, bearing in mind his antibody status and the possibility that cytotoxic immunosuppressive therapy for the leukaemia may result in progression to AIDS.